Modern Concepts of Cardiovascular Disease

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THE RECOGNITION OF PERICARDIAL DISEASE*

Some time ago, a patient was admitted to the hospital with severe ascites, requiring repeated paracenteses. Elsewhere a diagnosis of alcoholic cirrhosis of the liver had been made and a shunt operation performed without benefit. He admitted being an alcoholic, but it was evident from a preliminary examination that primary liver disease could not be the cause of his difficulties, since the pressure in the cervical veins was markedly elevated. Complete studies showed the correct diagnosis to be constrictive pericardial disease, and surgery effected a cure. The previous operative procedure had been unnecessary, based as it was on an incorrect diagnosis.

This example may be an extreme one, but it is illustrative of the problems arising in the diagnosis of pericardial disease. Sometimes they are relatively easy to solve, as in the instance quoted, but there are others that are difficult even for the expert. It is important to keep pericardial disease in mind as a possibility in all cases of presumed myocardial insufficiency and when congestive phenomena suggest cirrhosis of the liver or other intra-abdominal pathology. Though serious isolated pericardial involvement is relatively infrequent, the chance that a cure may be effected should be a constant spur to painstaking diagnostic alertness and effort.

Pericardial disease may lead to three different clinical syndromes: In the first, there is precordial pain of such a type and severity as to mimic myocardial infarction or other serious intrathoracic abnormality; this syndrome is the result of acute inflammation of the pericardium. In the second, a large cardiac outline by physical examination or x-ray, with or without manifestations of congestion throughout the body, suggests myocardial dilatation. In these cases, effusion into the pericardium is responsible for the clinical findings. If the effusion appears acutely, as in traumatic hemopericardium, a shock-like state may occur (one type of cardiogenic shock). In the third, a chronic adhesive process may interfere with cardiac filling and produce generalized chronic passive congestion, but in contrast to the tamponade of large effusions, the heart shadow is often only slightly or moderately enlarged. It should be noted that pericardial disease is often associated with serious pathology in the myocardium or endocardium, thus compounding the diagnostic difficulties. On the other hand, it is frequently manifest in a clinically isolated form, and in these instances dramatic cures may be achieved.

A review of the clinical signs and tools available to the physician in the diagnosis of the various pericardial syndromes seems warranted. Some of the signs readily detected at the bedside have been neglected by many physicians, and the significance of others has been more clearly elucidated by newer knowledge of pathophysiology and clinical experience. In this field, as in many others, instrumental means of diagnosis often have been overemphasized or too readily relied upon, despite the fact that experts have repeatedly stressed the limitations involved in their use and in the interpretation of graphs or films.

PHYSICAL EXAMINATION

Acute Pericarditis

The only physical sign of importance in the diagnosis of acute pericarditis is the pericardial friction rub. This is usually best heard in the midprecordial region, although the area of maximum intensity often varies from time to time and with the change of position of the patient. It may be systolic only, especially early in the disease, but characteristically it is a to-and-fro systolic and diastolic friction sound and may be mistaken for a double aortic murmur. In contrast to the latter, it sounds close to the ear and is increased in intensity by stethoscope pressure against the chest wall. The friction sound is often evanescent and may be missed if frequent examinations are not made.

Evidence of disease elsewhere in the body, *e.g.*, pneumonia, tuberculosis, malignancy, uremia, et

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cetera, suggests that acute pericarditis is a part of such diseases, either as a result of direct extension, by metastasis, or as a manifestation of a metabolic aberration. An apparently isolated acute pericarditis in a patient complaining of severe chest pain suggests either myocardial infarction or acute nonspecific pericarditis as the most likely possibilities. In acute pericarditis, the friction rub is usually present when the physician first sees the patient because of his pain, whereas in the case of infarction it does not appear until 24 to 48 hours have elapsed. Electrocardiographic examinations and transaminase determinations are helpful also in this differential diagnosis.

Pericardial Effusion

Some of the important physical findings in pericardial effusion have been listed in Table I and compared with those found in severe congestive failure and in constrictive pericarditis. Auenbrugger's sign (a sharp change in percussion note from resonant to dull or flat) has not been included because in the author's experience it has not proved to be of great value.

It should be noted that a visible and palpable apex beat may be found even though a large effusion is present. This may be due to predominant localization of the fluid laterally and pos-

Table I

Important Signs in the Differential Diagnosis of Pericardial Disease

Finding	Severe myocardial insufficiency	Pericardial effusion (large)	Constrictive pericarditis
	Clinical Signs		
Increased cervical venous pressure with a further rise on abdominal compression	Yes	Yes	Yes
Inspiratory distention of neck veins	Unusual	Yes	Yes
Palpable apex beat	Usually readily palpable	Absent often, but not always	Absent
Systolic rightward movement of chest wall	No, except with tricuspid regurgitation	No	Yes
Diastolic precordial thrust	No, except with tricuspid regurgitation	No	Yes
Protodiastolic third sound	Yes	Occasionally present	Yes
"Paradoxical" pulse $>$ 10 mm. Hg	Often present, especially with emphysema	Often present	Often present
"Paradoxical" pulse plus inspiratory distention of cervical veins	Rare	Often present	Often present
Ewart's sign	Rare	Often present	No
	Radiological Signs		
"Water bottle" contour on x-ray	No	Often present	No
Large nondescript heart shadow	Yes	Often present	Occasional
Pulsations of heart walls on fluoroscopy	Often diminished	Diminished	Diminished or increased
Pericardial calcification	No	No	In 50% of cases

teriorly, leaving the heart itself close to the chest wall. A similar mechanism may be responsible for the occasional audibility of a pericardial friction rub, despite the presence of an effusion. When an apex beat is either visible or palpable, the position of this beat relative to the left border of cardiac dullness should be noted with care. Characteristically, the former is located well within the latter boundary in the presence of a large effusion, and when elicited constitutes one of the most valuable physical signs of effusion. Muffling of the heart tones, on the other hand, is seldom sufficiently definite to be relied upon. The protodiastolic gallop rhythm occasionally heard with pericardial effusion has no diagnostic value or serious prognostic significance, as does the gallop associated with a dilated heart in the absence of pericardial disease.

A considerable percentage of patients with large pericardial effusions are found to have dullness, bronchial breath sounds, and bronchophony or egophony at the left lung base posteriorly. This finding, commonly referred to as Ewart's sign, has been provocative of much debate, since the mechanism of its production remains obscure. It probably is related to a compression atelectasis of the left lower lobe, but also it has been suggested that it could be due to rheumatic pneumonitis. In the experience of many observers, including the author, this sign is found in tuberculous pericardial effusions no less frequently than in cases of rheumatic etiology. Whatever the mechanism, the importance of the sign is obvious since the physician who is not alert may believe that the primary or only pathology is in the lung.

The interference with cardiac filling which results from the presence of a large effusion (cardiac tamponade) is productive of interesting and important signs. A serious degree of tamponade may result from as little as 300 to 400 ml. of fluid in the pericardium when the effusion develops acutely. On the other hand, in chronic effusion the pericardium usually becomes stretched in order to accommodate much larger quantities before serious interference with diastolic filling takes place. One of the major manifestations of such interference is a rise in venous pressure, best detected by a manometer reading from an antecubital vein, but also estimated with a fair degree of accuracy in the neck veins with the patient relaxed in a semiupright position (method of Sir Thomas Lewis). Further rise in venous pressure or filling of neck veins will occur when the examiner exerts firm pressure over the abdomen (sometimes called the hepatojugular reflux) because the heart is unable to accept the suddenly increased volume of blood in the already filled thoracic cava. These signs are not pathognomonic of pericardial effusion, of course, since they are found also in myocardial insufficiency, in constrictive pericarditis, and with space-taking lesions of the mediastinum. Their detection, however, should always remind the physician to include pericardial disease in his diagnostic considerations, and in a case of known pericardial effusion they are valuable guides in estimation of the degree of cardiac tamponade, provided shock is not also present.

In many cases of pericardial effusion, a considerable drop in systemic arterial pressure (greater than 10 mm. of mercury) occurs during the inspiratory phase of respiration. This phenomenon was first described by Kussmaul and is called the the "pulsus paradoxus." The name is a misnomer, however, since there is normally a small drop in systemic arterial pressure during inspiration, and the sign is simply an exaggeration of this normal cyclic change.[1] Furthermore, the sign is by no means limited to pericardial disease since it is frequently noted in patients with obstructive lower respiratory tract lesions who develop highly negative intrathoracic pressure during inspiration. The clue to the latter situation may be had by noting that expiration leads to distention of the neck veins. This is in contrast to pericardial disease in which *inspiratory* distention of these veins is noted.

Here, then, is a situation in which the coexistence of two signs, *i.e.*, "paradoxical" pulse plus inspiratory cervical vein distention, increases considerably the diagnostic significance which can be attached to either of the signs occurring alone. It should be further noted that in the important differential between pericardial effusion and severe myocardial insufficiency, this combination of signs is unusual in the latter and points strongly toward the former.

Constrictive Pericarditis

Some years ago, Dr. Claude Beck proposed a triad for the diagnosis of constrictive pericarditis,[2] including: (1) high venous pressure; (2) a small, quiet heart; and (3) ascites. This triad has been of considerable value, but it has given the general impression that the heart is always small in this condition. Such is not the case, there being some degree of enlargement, usually slight to moderate, in over 50 per cent of the cases[3] and at times it may be massive when a valvular lesion coexists or when there is an encapsulated residual pericardial effusion.[4] Perhaps the Beck triad could be better worded by saying that the "heart is smaller and quieter than would be expected for the degree of venous pressure elevation and congestive phenomena," but this would still fail to include those cases with marked enlargement (5.7 per cent in one series).[3]

The patient with constrictive pericarditis has the same signs related to impairment of diastolic filling as does a patient with a large effusion. Thus, elevation of systemic venous pressure, inspiratory distention of the neck veins, and a "paradoxical" pulse are observed. When this combination of signs occurs in association with a heart that is not massively enlarged, constrictive

pericarditis is a strong possibility. If a presystolic or protodiastolic third heart sound is heard, this also is in keeping with the diagnosis and should not lead the physician to conclude that the myocardium is damaged. When a protodiastolic third sound is present, it is due apparently to a checkrein mechanism, since electrokymographic and right ventricular cavity pressure curves show it to be coincident with the abrupt halt in ventricular filling occurring in early diastole.[5] A similar checkrein mechanism is undoubtedly responsible for the protodiastolic sound heard in endocardial fibroelastosis and in cardiac amyloidosis.

Of the various chest wall pulsatory phenomena that have been described as occurring in constrictive pericardial disease, the one of real diagnostic significance is the diastolic precordial thrust described originally by Skoda,[6] and emphasized in recent years by Dressler,[7] and Wood, *et al.*[8] Skoda pointed out that during systole, a precordial depression associated with a rightward propulsion of the chest wall occurs. This is followed, during diastole, by a more impressive precordial thrust which may be mistaken for an apex beat by one who is careless in timing cardiac events. A somewhat similar pulsatory situation occurs in tricuspid regurgitation, but in that case the rightward propulsion during systole is more prominent, and the diastolic leftward and outward movement less so. Dressler attributes the pulsatory phenomena occurring in constrictive pericardial disease to a change in the mechanism of volumetric diminution of the heart, whereby the marginal movements of the ventricular walls prevail over the usually predominant longitudinal shortening of the ventricular cone. Wood, *et al.*, call this the diastolic heart beat and show it to be coincident with the protodiastolic third sound when this is present. They attribute it to expansion of a "window," or a weak spot, in the pericardial scar. It is a phenomenon which does not require the presence of any adhesions external to the heart for its production.

INSTRUMENTAL DIAGNOSTIC AIDS

Electrocardiogram

The electrocardiogram is particularly valuable in the diagnosis of acute pericarditis. The characteristic change in the S–T segment (upward deviation in the standard and precordial leads) is due to the current of injury flowing in the superficial layers of the myocardium affected by the adjacent inflamed pericardium. The direction of the deviation is concordant, in contrast to the discordant deviation which is usual in myocardial infarction. Furthermore, in pericarditis there tends to be a persistent downstroke of the QRS complex at the junction with the S–T segment, in contrast to the usual absence of this stunted S wave in myocardial infarction.[9] In chronic pericarditis, T-wave inversion in all leads except V_R is the rule.

In pericardial effusion and constrictive pericarditis, the complexes tend to be small and the T waves flattened, but these are nonspecific changes. Arrhythmias, including atrial fibrillation, are common in constrictive pericarditis.

Roentgenological Examination

The roentgenogram is of no value in acute pericarditis; it may be of considerable consequence in pericardial effusion and in constrictive pericarditis. In the former, the characteristic "water bottle" or "gourd" configuration may be noted, particularly if the parietal pericardium has not lost its elasticity. In many instances, however, the fluid distended pericardium is an atonic sac and is indistinguishable on the ordinary film from an atonic, dilated heart. Change in contour with change of body position may likewise be of little help, since a failing heart often changes in like manner. An important and valuable clue is the combination of a large heart shadow with clear lung fields and normal vascular markings.[4]

The pulsatory phenomena seen on fluoroscopic examination frequently prove misleading, since the badly damaged myocardium may show the same feeble pulsations that are to be expected in pericardial effusion.

For these reasons, angiocardiography is being used to distinguish between pericardial effusion and myocardial dilatation. By this means the margin of the contrast substance in the right atrium is observed and its position compared with the right border of the cardiac shadow. Assuming the wall of the right atrium to be no more than several millimeters in thickness, a discrepancy greater than this can be attributed to thickening or fluid in either the pericardium or the right mediastinal pleura. Both positive contrast (diodrast, urokon, et cetera), and negative contrast (carbon dioxide*), have been used. The author and his co-workers prefer the latter because of the simplicity of the procedure, the lack of reaction, and the better visualization often obtained.[10]

In constrictive disease, the roentgenogram shows calcification of the pericardium in about half the cases. Absence of calcification cannot be relied upon, therefore, to eliminate the diagnosis, and it should be noted, furthermore, that not all cases of calcific pericarditis are of clinical importance. Angiocardiography, either positive or negative contrast, is of value in showing deformity of the right atrial wall.[10, 11]

Fluoroscopic examination occasionally shows increased rather than decreased marginal pulsations of the left cardiac border,[4] but when this is true, contrasting diminished pulsations of the great vessels are noted.[12]

Pure carbon dioxide must be used. See technique described in reference #10.

Other Instrumental Aids

The right ventricular pressure curve obtained by catheterization is often very helpful diagnostically in constrictive pericarditis. By this means, there is demonstrated an early diastolic dip followed by a plateau.[5] The latter corresponds, except for some lag in onset, with the diastolic plateau of ventricular border movement.

The electrokymogram also often shows a characteristic pattern for the ventricular border movements.[13] It has been described as the "flat-top and V" curve. The "flat top" refers to the plateau produced by the impediment to diastolic filling. The downward limb of the "V" is inscribed by the inward movement of the ventricular contraction, and the steeper upward limb represents the outward recoil with early diastolic filling (often associated with a readily visible collapse of the neck veins).

PERICARDIOCENTESIS

Final proof of the presence of pericardial effusion is obtained, of course, by needle aspiration of fluid from the sac. This procedure should not be undertaken lightly, however, since there is a definite risk involved. Fatal tears of the myocardium have occurred, a coronary or internal mammary artery may be injured, or there may be induced reflexly a state of shock or cardiac arrhythmia. The patient should be prepared by administration of a narcotic, plus atropine, to lessen the chance of the last two of these. Several routes have been advocated for the introduction of the needle, and the apical is particularly safe when the apex beat is detected well within the left border of cardiac dullness. The author, however, generally prefers the subxiphoid approach, the patient lying semiupright with the spine supported in marked extension by pillows. The needle should be made the exploratory electrode of an electrocardiographic machine by attachment to a precordial lead wire, the leads to the central terminal being attached in the usual fashion. An assistant may then note immediately the happenstance of needle contact with the myocardium, since instant displacement of the S–T segment will occur. When the fluid has been aspirated there is often an indication for the introduction of air into the sac, since this may provide information roentgenologically as to the thickness of the parietal layer, the presence or absence of adhesions and loculi, and the actual size of the heart. It may also be of therapeutic value in that it is believed to inhibit the development of an adhesive process.

ETIOLOGICAL DIAGNOSIS

The complete diagnosis of pericardial disease is not made, of course, until the cause of the process has been established. This is by no means always possible, but every attempt should be made to accomplish it, since this is the key to proper therapy. It is particularly important to subject any fluid obtained by aspiration to complete bacteriological and cytological examination. The frequency with which disseminated lupus erythematosus is responsible for pericardial disease should not be overlooked. Tuberculosis is particularly difficult to affirm or disprove as a cause, and the tubercle bacillus should be sought for by the aid of a complete bacteriological study.* A repeatedly negative tuberculin skin test has great value in excluding this diagnosis.

The importance of etiological diagnosis has led to more and more utilization of open biopsy of the pericardium, particularly when there is chronic inflammation. The risk is almost negligible with modern surgical techniques, and the likelihood of a complete diagnosis being made is good. It also may be of therapeutic value since a pericardial window may be established to allow the effusion to drain into the adjacent pleura and to obviate the need for repeated pericardial taps.[14]

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*L. E. cells may be found in the pericardial fluid—Editor.

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